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NOT TO BE PUBLISHED IN THE OFFICIAL REPORTS

IN THE COURT OF APPEAL OF THE STATE OF CALIFORNIA

SECOND APPELLATE DISTRICT

DIVISION FIVE

DIANE OAKES, Plaintiff and Appellant, v. WYETH, Defendant and Respondent.	B199799 (Los Angeles County Super. Ct. No. DD000894 JCCP4032)

APPEAL from a judgment of the Superior Court of Los Angeles County, Daniel S. Pratt, Judge. Reversed.

The Elstead Law Firm, John Clifton Elstead; Esner, Chang & Ellis and Andrew N. Chang for Plaintiff and Appellant.

Gordon & Rees, Stuart M. Gordon, Fletcher C. Alford, Kai Peters, Ryan B. Polk; Arnold & Porter and Ellen K. Reisman for Defendant and Respondent.

I. INTRODUCTION

Plaintiff, Diane Oakes, appeals from a summary judgment entered in favor of defendant, Wyeth, formerly known as American Home Products Corporation. Plaintiff's complaint arose from her use of defendant's diet drug, fenfluramine. The trial court granted defendant's summary judgment motion on the ground plaintiff could not establish a causal connection between her consumption of fenfluramine and three heart conditions: pulmonary hypertension; valvular heart disease; and coronary artery disease. We reverse.

II. BACKGROUND

A. Plaintiff's Complaint

On February 9, 2000, plaintiff filed her complaint for damages for permanent injuries to her heart which she allegedly suffered as a result of consuming fenfluramine. Plaintiff alleged causes of action for strict liability, negligence, warranty breach, deceit, misrepresentation, consortium loss, and violations of Business and Professions Code sections 17200 and 17500.

B. Conflicting Medical Testimony

On December 5, 2006, defendant moved for summary judgment on the ground plaintiff had no admissible evidence to prove her injuries were proximately caused by her use of the diet drug, Pondimin, which is the trade name of the generic drug fenfluramine. The following evidence was produced in support of and in opposition to the summary judgment motion. Plaintiff is in her sixties. Plaintiff used fenfluramine and phentermine on a daily basis from February 1996 to May 1997. Phentermine is a sympathomimetic amine which allegedly acts like amphetamines and was marketed as an appetite

suppressant. In October 1998, plaintiff developed an upper respiratory viral-like illness with laryngitis that lasted for about six weeks. In February 1999, plaintiff began experiencing swelling and fluid retention in her lower legs and feet (pedal edema) and severe shortness of breath.

In March 1999, Dr. Richard W. Terry, plaintiff's treating cardiologist at that time, arranged for her to have an echocardiogram. The echocardiogram showed among other things: a normal aortic valve with no aortic insufficiency; mitral valve normal by echo; moderate mitral regurgitation; and normal tricuspid valve. With respect to the tricuspid valve, Dr. Terry clarified at his deposition: "Normal by appearance; in other words, not thickened, not prolapsing, et cetera. But it certainly leaked severely, so it was not normal." However, the valvular structure was normal. In response to whether plaintiff had valvular heart disease, Dr. Terry testified, "It's a horse-and-cart issue; in other words, did the valve problem cause the heart failure or did the heart failure cause the valve problem?" In a report dated March 31, 1999, Dr. Terry diagnosed plaintiff with "[d]evelopment of progressive congestive [heart] failure, which is predominantly right heart failure" which began around mid-February 1999. Dr. Terry attributed plaintiff's condition to "a very striking and persisting viral illness raising a strong possibility [there was] a myocarditis" which is an inflammatory disease of the myocardium or heart muscle. Dr. Terry thought that the primary problem was cardiomyopathy, which is the malfunctioning of the left ventricular pumping chamber. Dr. Terry considered the valvular abnormalities were secondary to cardiomyopathy. Dr. Terry also noted that there was "'[a]nother definite possibility'" of hypertensive heart disease but that he would have expected more hypertrophy and potentially dilatation if there was a chronic finding rather than a more labile event aggravated by plaintiff's current illness. Dr. Terry also noted a potential for ischemic disease because of the regional abnormality. Dr. Terry did not pursue the ischemic disease issue at that time. Dr. Terry also noted plaintiff had moderate mitral insufficiency with severe tricuspid insufficiency and pulmonary hypertension which he related to the cardiomyopathy. Dr. Terry anticipated plaintiff's mitral and tricuspid regurgitation and pulmonary hypertension would resolve if her

cardiomyopathy resolved. Dr. Terry also considered factors such as plaintiff's blood pressure and obesity.

Dr. Terry began treating plaintiff with medications to: get rid of fluid buildup; lower her blood pressure; dilate her arteries; make it easier for her heart to pump blood forward; and improve the weakened heart muscle performance. At an office visit on April 29, 1999, plaintiff no longer had shortness of breath or edema. Plaintiff had an excellent response and heart function was approaching normal. An echocardiogram taken in September 1999 showed "mild mitral regurgitation" and her mitral, aortic, and tricuspid valves were normal. Dr. Terry noted as of September 23, 1999, plaintiff had made a "'fantastic recovery.'" Dr. Terry described the basis for his opinion, "[B]ased on the full echo report, because of the virtual complete recovery of her heart function to normal and the resolution of any significant valve leakage." Dr. Terry testified that many people have a tricuspid regurgitation of a mild degree.

In a February 16, 2000 report, Dr. Terry stated that plaintiff had no cardiac symptoms. She was not complaining of shortness of breath, swelling, waking up short of breath, or chest pains. Dr. Terry reported that on an April 24, 2000 echocardiogram plaintiff's aortic valve was structurally normal with normal flows and velocities. Plaintiff's mitral valve was structurally normal with mitral regurgitation. Dr. Terry testified he would not get "particularly excited" about mitral regurgitation which is the same as "trace" because the mitral valve will occasionally leak. Dr. Terry could not assess whether plaintiff had pulmonary hypertension because the leak was so trivial the velocity of the tricuspid valve was not obtained.

Dr. Terry never concluded that plaintiff's mitral regurgitation was due to her fenfluramine use. He never changed his mind that the cardiomyopathy resulted in the: mitral regurgitation; the tricuspid regurgitation; and the pulmonary hypertension. Dr. Terry never made any determination that her cardiomyopathy was due to plaintiff's fenfluramine use. Dr. Terry was unaware of any association between cardiomyopathy and fenfluramine. Dr. Terry last treated plaintiff on May 3, 2000. Plaintiff did not return for follow-up care with Dr. Terry after that date.

Dr. Sharon Hunt, a Stanford University cardiologist, examined plaintiff in November 2000. Dr. Hunt reviewed an echocardiogram performed on plaintiff on December 6, 2000. Dr. Hunt noted it was basically normal meaning '"minor apical hypokinesis, overall normal left ventricular function'"; implying that she discounted the minor abnormality. Dr. Hunt testified: "The term hypokinesis describes lack of perfectly normal contraction or kinesis with the heartbeat. The term apical refers to the apex or tip of the heart." Dr. Hunt did not determine "any potential cause" of the apical hypokinesis. Dr. Hunt did not determine that diet drugs were the cause of plaintiff's apical hypokinesis. Dr. Hunt also noted there was minor mild mitral regurgitation and tricuspid regurgitation both of which are very common and felt to be physiologic and of no significance. Dr. Hunt testified the December 6, 2000 echocardiogram showed: plaintiff did not have valvular heart disease; plaintiff had a normal mitral valve with mild mitral regurgitation; a normal aortic valve in structure and function with no regurgitation; and the right ventricular systolic pressure was within normal limits, which would exclude any pulmonary hypertension. Dr. Hunt also noted that two prior echocardiograms showed that plaintiff did not have pulmonary hypertension. While providing treatment, Dr. Hunt did not address the issue of whether diet drug use had anything to do with plaintiff's cardiovascular problems. Dr. Hunt believed the cardiomyopathy was idiopathic in nature meaning an unknown cause. Dr. Hunt determined the cardiomyopathy was idiopathic by excluding underlying metabolic diseases such as thyroid disease or anemia. Dr. Hunt excluded valvular disease and tried to exclude ischemic disease (blocked arteries).

Dr. Edward Woods began treating plaintiff in August 2003. Dr. Woods is a cardiothoracic surgeon, who practices in Pennsylvania. Plaintiff is the only patient that Dr. Woods has diagnosed with coronary artery disease due to fenfluramine use. Dr. Woods referred plaintiff to a cardiologist, Dr. Karandeep Singh. Dr. Woods and Dr. Singh practice together. Dr. Singh performed a cardiac catheterization and coronary angiography on plaintiff. Dr. Singh found a blockage of the left anterior descending artery. According to Dr. Singh, one cannot tell what causes the blockage. Rather, Dr. Singh testified: "You make an assumption that in most cases that blockage is from

buildup of plaque, which is a mixture of cholesterol [and] calcium, but based on the picture that's only an assumption. [¶] You can't tell more than that." The blockage could also be the result of dissection or a tear, trauma, and inflammation. Dr. Singh did not make a determination as to the most likely cause of the heart blockage because it was not relevant to plaintiff's care. Dr. Singh did not ever consider fenfluramine as a possible cause of the blockage because it would not have changed what he needed to do for plaintiff at the time he saw her. He was unaware of any literature that led him to believe fenfluramine caused blockages of coronary arteries. Dr. Singh concluded that all the pressures inside the heart were normal including for the pulmonary artery. Dr. Singh concluded that plaintiff did not have pulmonary hypertension. Dr. Singh concluded the blockage in the left anterior descending artery caused an inferoapical akinesis or a wall motion abnormality. Dr. Singh determined that plaintiff had cardiomyopathy by her medical history from California. Dr. Singh indicated that he did not know what caused the blockage but that plaintiff had several risk factors such as: high cholesterol; diabetes; high blood pressure; obesity; and she was post-menopausal. Dr. Singh did not consider fenfluramine to be a cause of plaintiff's coronary artery disease.

Dr. Richard Butcher is a cardiologist who practices with Dr. Wood. Dr. Butcher treated plaintiff on Dr. Wood's referral. Dr. Butcher read an exercise echocardiogram and performed a transesophageal echocardiogram on plaintiff. Dr. Butcher concluded that the anatomy of the mitral aortic and tricuspid valves was normal. He made no determination as to the pulmonic valve because it was not well seen. Dr. Butcher stated in his report that there was: mild micro regurgitation; no aortic regurgitation; and moderate tricuspid regurgitation. There was no pathologic regurgitation of the pulmonic valve. Dr. Butcher concluded there was no valvular heart disease related to anorectic drugs. Dr. Butcher testified: "The findings that are attributed to anorectic drugs are several and she didn't appear to have any of them to me. One of the findings is that an abnormal thickening of the valve leaflets, usually focal, and another is more severe aortic regurgitation than is mentioned in this report and another is aortic regurgitation, which was absent in this case. . . . [¶] The criteria for diagnosing valvulopathy from anorectic

drugs is two plus or more micro regurgitation and one plus or more aortic regurgitation plus or minus abnormal thickening of the valve leaflets of either of those two valves. The anorectic drugs are not known to cause an increase in tricuspid regurgitation." The size of the left ventricular cavity was not enlarged and was pumping a volume of blood in the normal range. Dr. Butcher testified at his deposition there was no evidence plaintiff had pulmonary hypertension. Dr. Butcher concluded plaintiff's wall motion abnormalities were consistent with distal anterior descending coronary artery disease. Dr. Butcher would not list the drug fenfluramine as a risk factor for coronary artery disease. Dr. Butcher was unaware of any association between fenfluramine and coronary artery disease. Dr. Butcher believed that there was no increased incidence of tricuspid regurgitation associated with anorectic drugs.

Dr. Woods repaired the blockage in October 2003. At his deposition, Dr. Woods testified he believed that plaintiff's cardiomyopathy was caused by her diet drug use. However, he did not know the mechanism by which diet drugs could cause cardiomyopathy. Dr. Thomas Gregory Quinn, a cardiologist, began treating plaintiff in October 2003 after her surgery. There was no information to suggest that plaintiff had valvular heart disease. Dr. Quinn did not determine that plaintiff had pulmonary hypertension.

C. Defendant's Analysis And Dr. Robert Kraus's Declaration

In support of the summary judgment motion, defendant argued: "Plaintiff brings this action alleging that defendant's diet drug, Pondimin, caused plaintiff to experience congestive heart failure and coronary artery disease. However, there is absolutely no admissible scientific evidence of any causal link between diet drugs and congestive heart failure or coronary artery disease. Indeed, all of the five cardiologists who have treated plaintiff agree that she has no diet drug induced cardiovascular disease of any kind." (Italics and bold face omitted.) Defendant contended the following facts were undisputed. Five cardiologists treated plaintiff. All five cardiologists agreed that there

was no medical evidence that plaintiff has any diet drug induced cardiovascular disease. Dr. Woods, is a surgeon and not a cardiologist. Dr. Woods has no experience diagnosing or treating diet drug patients. Plaintiff had cardiomyopathy/congestive heart failure and a blocked coronary artery. There are no published medical studies, journal articles, or texts showing any kind of association between diet drugs and cardiomyopathy or coronary artery blockage. There is no known mechanism of action by which diet drugs could cause cardiomyopathy or blocked coronary arteries. The view that diet drugs cause cardiomyopathy or blocked coronary arteries is not generally accepted in the cardiology community. Dr. Woods cannot identify any physician other than himself who holds that view. Dr. Woods has never tested his theory that diet drugs cause cardiomyopathy and coronary artery blockage. Dr. Woods does not know the mechanism by which diet drugs allegedly cause cardiomyopathy or coronary artery disease.

Further, defendant relied on Dr. Kraus's declaration. Dr. Kraus declared he was retained by defendant as a consultant. Dr. Kraus is a practicing cardiologist and board certified in internal medicine and cardiology. In reaching opinions about whether a drug or substance can cause cardiovascular disease, cardiologists rely upon epidemiological studies and other resources such as published research, medical texts, and peer-reviewed medical journal articles. Dr. Kraus declared that he was thoroughly familiar with the medical literature and published studies on the effects of diet drugs on the cardiovascular system. Dr. Kraus defined cardiology as the medical specialty devoted to diagnosing, determining the causes of, and treating cardiovascular diseases. Dr. Kraus defined "dilated cardiomyopathy" as a condition in which the heart's muscle fails to adequately contract. Dr. Kraus declared: "One of the most common causes of dilated cardiomyopathy is a viral infection that spreads to the heart. [¶] [] . . . If dilated cardiomyopathy becomes sufficiently severe, it can lead to congestive heart failure. Congestive heart failure is a term used to describe a constellation of symptoms that develop when the heart is unable to pump enough blood to supply the body's needs. As a result, blood 'backs up' (becomes 'congested') behind the heart, leading to an accumulation of fluid in the lungs and body tissues. Thus, one common symptom of

congestive heart failure is fluid retention and swelling in the feet and lower legs ('pedal edema')."

Dr. Kraus examined plaintiff's medical records and noted the following. Plaintiff developed a significant viral illness in October 1998 which lasted for several months. In February 1999, plaintiff began experiencing what Dr. Kraus described as classic signs of congestive heart failure—swelling and fluid retention in her lower legs and feet and severe shortness of breath. In March 1999, plaintiff's treating cardiologist at that time, Dr. Terry, diagnosed plaintiff as suffering from congestive heart failure due to dilated cardiomyopathy, which was likely viral in origin. Dr. Kraus concurred with Dr. Terry's diagnosis. Dr. Kraus did not know of any studies or professional literature which showed any association between diet drugs and cardiomyopathy. According to Dr. Kraus, "[I]t has never been shown that diet drugs have any effect upon the heart's muscle." Dr. Kraus further declared, "[T]here is no known mechanism of action by which diet drugs could cause cardiomyopathy or other diseases of the heart's muscle." Dr. Kraus stated that it was not generally accepted among cardiologists that diet drugs are associated with cardiomyopathy. Dr. Kraus was not aware of any cardiologist who held the opinion that there was an association between diet drugs and cardiomyopathy.

Dr. Kraus examined an echocardiogram performed on plaintiff while she was treating with Dr. Terry. Dr. Kraus also reviewed a transcript of Dr. Terry's deposition and the results of the echocardiogram. The echocardiogram showed regurgitation of the mitral and tricuspid valves. The echocardiogram also showed that plaintiff's heart valves were structurally normal. Dr. Kraus declared that "none of the thickening that has been associated with alleged diet drug induced valve disease" was present. Dr. Kraus concurred with Dr. Terry's conclusion that plaintiff's valvular regurgitation was secondary to her cardiomyopathy. It was not an independent disease process related to diet drugs.

Dr. Kraus also noted that plaintiff's echocardiogram taken by Dr. Terry showed she had pulmonary hypertension. Dr. Kraus concurred with Dr. Terry's deposition testimony that the pulmonary hypertension was not an independent "primary" disease

process or caused by diet drugs. Rather, the pulmonary hypertension was secondary to the cardiomyopathy.

Dr. Terry treated plaintiff's cardiomyopathy with drugs to: reduce fluid retention; lower blood pressure; dilate arteries; reduce the thickening of the heart muscle; and strengthen heart muscle contraction. Dr. Kraus declared: "The effects of that treatment were rapid and dramatic. Plaintiff's cardiomyopathy quickly resolved; and simultaneously therewith, her valvular function and cardiac pressure also returned to within normal limits. The fact that valvular function and cardiac pressure returned to within normal limits when the cardiomyopathy was treated shows very clearly that those conditions were secondary to the cardiomyopathy and were not independent disease processes caused by diet drugs. Had the valvular regurgitation or pulmonary hypertension been independent disease processes caused by diet drugs, the treatment regimen instituted by Dr. Terry would have caused little or no improvement of those conditions."

Dr. Kraus concurred with Dr. Hunt that there was no evidence that plaintiff was suffering from valvular heart disease, primary pulmonary hypertension, or any other cardiovascular conditions caused by diet drugs. Dr. Kraus also examined medical records and deposition testimony of two other cardiologists, Dr. Singh and Dr. Butcher. Dr. Kraus concurred with Dr. Singh's deposition testimony that diet drugs are not a cause of coronary artery disease. Dr. Kraus also agreed with Dr. Singh that diet drugs did not cause plaintiff's coronary artery disease. Dr. Kraus declared: "There are no published studies or other medical literature showing any association between diet drugs and coronary artery blockage. Nor is there any known mechanism of action by which diet drugs could cause a blockage of a coronary artery. It is not generally accepted among cardiologists that diet drugs are associated with coronary artery disease. Indeed, I am not aware of any cardiologist who holds the opinion that there is such an association. Typically, such blockages are caused by deposits of cholesterol and/or calcium. Plaintiff's medical records indicate that she has a number of risk factors—completely unrelated to diet drug use—that place her at exceedingly high risk for coronary artery

blockage, including high cholesterol, a sedentary lifestyle, high blood pressure, pre-diabetes/diabetes, obesity, and her post-menopausal status."

D. Plaintiff's Opposition And Dr. Wood's Declaration

In opposition to the summary judgment motion, plaintiff argued that defendant's evidence did not conclusively negate her claims that her use of the diet drugs caused her subsequent heart disease. Plaintiff contended that defendant had taken the deposition testimony of her five treating cardiologists out of context and distorted the testimony insofar as it related to her heart disease. Plaintiff disagreed with defendant's undisputed fact statement and claimed that none of the five cardiologists were ever asked nor did they agree that plaintiff's cardiovascular diseases were not caused by diet drugs. Plaintiff contended the five cardiologists were not asked questions establishing an expertise with diet drugs such that defendant cannot attribute an opinion to the cardiologists on this issue. Plaintiff further argued: Dr. Terry described plaintiff's heart problem in 1999 as cardiomyopathy or heart failure; the 1999 echocardiogram showed three accepted signs of diet drug-induced heart disease; the three signs were severe pulmonary hypertension, severe tricuspid valve insufficiency and moderate mitral valve insufficiency; Dr. Terry answered "no" when asked if he concluded that the cardiomyopathy and mitral valve insufficiency were due to diet drugs; Dr. Terry had no expertise with diet drugs and was never asked whether he did anything to determine diet drugs were not the cause; Dr. Terry did not know that plaintiff had used diet drugs; and Dr. Terry did not find plaintiff's blocked artery. Plaintiff pointed out that Dr. Terry testified: it was a "horse-and-cart issue . . . did the valve problem cause the heart failure or did the heart failure cause the valve problem?"; a virus was one of many "possibility[ies]" of the cardiomyopathy; plaintiff's valves were normal by echo appearance but they still leaked; he would expect the valvular insufficiencies due to a pulmonary hypertension to ease if the cardiomyopathy resolved; and drugs would make plaintiff feel better but not make her

heart healthier. Plaintiff then cited evidence from Dr. Hunt and Dr. Woods that if the drugs were discontinued the disease could come back.

As part of her opposition, plaintiff filed a declaration by Dr. Woods. Dr. Woods declared that he is a cardiothoracic surgeon board certified in thoracic surgery. Dr. Woods diagnoses and treats diseases of the: thoracic cavity; heart; vessels; lungs; esophagus; trachea; chest; and chest wall. There is a difference between the backgrounds of a cardiologist and a cardiothoracic surgeon. A cardiologist's background is in internal medicine. By contrast, a cardiothoracic surgeon's training involves general surgery. Dr. Woods has extensive experience in the diagnosis and treatment of cardiovascular diseases, including: valvular heart disease; pulmonary hypertension; and coronary artery disease. His experience includes interpretation of echocardiograms and other procedures designed to detect cardiovascular diseases.

Dr. Woods declared: "I have spent many hours reviewing medical literature and studies regarding diet drugs such as fenfluramine and their effects on the cardiovascular system. I am thoroughly familiar with their reasoning and findings. In arriving at my opinions as to whether diet drugs cause cardiovascular disease, I rely on those writings, as well as on other epidemiological studies, published research, medical texts, and peer-reviewed medical articles that cardiothoracic surgeons and cardiologists typically rely on. I also rely on my direct visualization of the cardiovascular system during surgery, which gives me direct knowledge of the limitations of echocardiograms. They provide only indirect visualization of the cardiovascular system and do not always reveal the presence of heart diseases, such as valvular heart disease, pulmonary hypertension, and coronary artery disease. A valve may look normal by echocardiographic criteria and still be abnormal and diseased. I see this day-in-and-day-out in surgery. Valves are abnormal but do not appear abnormal by echocardiographic criteria. Echocardiograms are relatively crude for evaluating these things and thickened valve leaflets do not always show up in echocardiograms."

Dr. Woods continued: "The opinions I state in my deposition and in this declaration regarding [plaintiff] and the causal association between diet drugs and

cardiovascular disease are all expressed to a reasonable degree of medical probability. They are based on my extensive experience as a heart surgeon, reviewed medical literature and studies, [plaintiff's] medical history and records, my care and treatment of her, and my direct visualization of her cardiovascular system during surgery. [¶] [] I reviewed the medical records and depositions of the [five] treating cardiologists . . . . Dr. Terry treated [plaintiff] in 1999 for what he described as cardiomyopathy or heart failure. His records show that she had severe pulmonary hypertension of nearly 50mmHg, severe tricuspid valve insufficiency, and moderate mitral valve insufficiency. A later echocardiogram also shows that she has aortic valve insufficiency. These are accepted symptoms of diet drug related heart disease and it is my opinion that the pulmonary hypertension and valvular insufficiencies were caused by extended diet drug use. Dr. Terry suggest that the cardiomyopathy was possibly by a virus, but I do not agree. I operated on [plaintiff] on October 16, 2003 and it is my opinion that her cardiomyopathy was not viral related. I looked at her heart and saw that her pericardium was completely clean and free and that there was no evidence of viral related pericarditis. I have operated on many people with histories of viral cardiomyopathy and this is not what you expect to see. There is no evidence of residual inflammation and scarring that you see when there has been viral related cardiomyopathy or heart failure."

Dr. Woods explained: "Dr. Terry said that he does not know whether the cardiomyopathy caused the pulmonary hypertension and valvular insufficiencies or vice versa, but it is my opinion that they were caused by extended diet drug use. Cardiomyopathy is a nonspecific term referring to improperly functioning ventricles that have dilated or stretched and do not expel their normal output of blood. Valvular insufficiencies can cause a ventricle to overwork and dilate and result in valvular insufficiency related cardiomyopathy. This is what happened with the left side of [plaintiff's] heart. Her severe tricuspid valve insufficiency shows that the right side of her heart was also affected by diet drugs and caused severe pulmonary hypertension. [Plaintiff] had no history or symptoms of cardiomyopathy, valvular insufficiency, or pulmonary hypertension before she used diet drugs and there is no other probable

medical explanation. She was not at risk for these diseases before she used diet drugs. Her parents' heart problems did not arise until they were in their late seventies and her cholesterol and blood pressure levels were too low to be risk factors. She was not diagnosed with diabetes until years after she stopped using diet drugs and she did not smoke enough to be significant. She was obese but, that too, was not significant enough to be a real risk factor."

Dr. Woods discussed the limitations of echo based technology in discerning valve related disease: "The echocardiograms performed by Dr. Terry and the other cardiologists who treated [plaintiff] do not show that her heart valves are in fact structurally normal and that she did not have and does not have valvular heart disease and pulmonary hypertension. Failure to echo detect structural abnormalities, such as thickening, does not mean that the valves are normal. I see this all the time in surgery, where the echo showed normal valves, but the valves are abnormal and diseased. The echo criteria for valves, compared to actually looking at them, is still relatively crude and inexact. Valves can be abnormal and diseased and not appear abnormal and diseased by echo criteria. Dr. Terry apparently recognized this when he said that [plaintiff's] valves appear normal by echo, but still leaked and were not normal."

Dr. Woods concluded plaintiff's heart disease was caused by extended diet drug usage: "It is my opinion that [plaintiff's] blocked left anterior descending artery . . . , which I bypassed on October 27, 2003, was caused by extended diet drug use. Dr. Krause says that it is not generally accepted that diet drugs cause coronary artery disease or blocked arteries, but is more accurate to say that there have been no studies specifically looking for such a cause. The drugs were withdrawn from the market before there was an opportunity for such studies. He also says that blocked arteries are typically caused by deposits of cholesterol, but I saw no evidence of this when I operated on [plaintiff]. I did not see any evidence of the plaquing and yellow fatty or calcium deposits associated with cholesterol related atherosclerosis that I have seen in hundreds of hearts I have operated on. I saw what looked to be fibrotic lesions like those seen in the valves of patients who used diet drugs and in the valves and arteries of patients with

carcinoid disease. [¶] [] The cardiac [catheterization] performed on August 28, 2003 showed that [plaintiff's] other coronary arteries were clean. This is significant. The [left anterior descending artery] is a small artery that branches off from the base of the aorta just above the aortic valve. The blood going through the aortic valve and aorta into the [left anterior descending artery] comes from the left atria and left ventricle, which normally receive blood from the right side of the heart after it has been filtered through the lungs. [Plaintiff] had a foramen ovale or small hole between the two sides of her heart that allowed unfiltered blood with diet drugs to bypass the lungs and go directly from the right side to the left side of her heart. This exposed the small [left anterior descending artery] to unfiltered blood and caused the blockage. The blockage was so significant that, had it existed before [plaintiff] used diet drugs, I would have expected significant symptoms, such as angina."

In terms of plaintiff's illness and her future, Dr. Woods declared: "I agree with Dr. Hunt that [plaintiff's] valvular heart disease and pulmonary hypertension have resolved or been compensated by drug therapy, but not cured. [Plaintiff] will have to remain on drug therapy for the rest of her life. Dr. Terry correctly noted that her valves still leak and later tests showed that her pulmonary pressures are still abnormal. Her pulmonary pressures were borderline when measured on August 18, 2003, but they were obtained late in the afternoon when she was dehydrated and had not been exercised. Had they been measured when she was hydrated and exercised, they would have been higher. Her systolic blood pressure was also lower than normal at the time and her pulmonary pressures [were] lower for the same reason. [¶] [] In sum, it is my opinion that [plaintiff's] cardiomyopathy, valvular insufficiencies , pulmonary hypertension, and blocked [left anterior descending artery] were caused by extended use of diet drugs. They have resolved or been compensated by drug therapy and surgery, not cured. The causal association between diet drugs, valvular insufficiencies, and pulmonary hypertension is widely accepted in the medical community and there are accepted medicals reasons to extend the association to coronary artery disease. Given that patients with carcinoid disease also have high levels of serotonin in their blood and suffer both

valvular and coronary artery disease, it is reasonable to conclude that diet drugs also [affect] arteries. Carcinoid studies show that serotonin is not valve specific and [affects] arteries as well."

Dr. Woods described the relevant medical literature: "Prior to arriving at my opinions, I reviewed many articles and studies dealing with diet drugs and carcinoid disease and their effects on the cardiovascular system. They show that the causal association between diet drugs and heart disease is widely accepted in the medical community and that resulting excess serotonin in the blood is the causative mechanism." Attached to Dr. Woods's declaration is a list of 27 articles that discuss the relationship between diet drugs and carcinoid disease and their effects on the cardiovascular system.

E. The Order Granting Defendant's Summary Judgment Motion

The trial court granted defendant's summary judgment motion. The trial court ruled: defendant met its initial burden of showing that plaintiff's heart problems were not caused by diet drugs through Dr. Kraus and "several experts" responsible for her care; defendant's five expert opinions established that there was no causation; and Dr. Woods's declaration did not create a triable issue of fact. The trial court ruled Dr. Woods's declaration was inconsistent with his deposition testimony that he did not know how plaintiff's diet drug use purportedly caused her injuries and he was unaware of any scientific articles supporting that conclusion. The trial court found: Dr. Woods's opinion that the blockage was due to a foramen ovale between the two sides of plaintiff's heart allowing unfiltered blood to pass through her heart was speculative; Dr. Woods would have expected significant symptoms to have occurred if that problem had existed before plaintiff used diet drugs; and Dr. Woods's disagreement with Dr. Terry's viral cardiomyopathy diagnosis did not create a triable issue of material fact because it did not establish that there was causation between the diet drugs and plaintiff's injuries. The trial court concluded, "In sum, the court is presented with the evidence of defendant's five expert opinions and the testimony of Dr. Woods submitted by plaintiff, to determine

whether the issue of causation is one that is materially disputed through competent, unspeculative expert testimony." After the trial court entered judgment against plaintiff, this timely appeal followed.

III. DISCUSSION

A. Standard of Review

In *Aguilar v. Atlantic Richfield Co.* (2001) 25 Cal.4th 826, 850-851, our Supreme Court described a moving party's summary judgment burdens as follows: "[F]rom commencement to conclusion, the party moving for summary judgment bears the burden of persuasion that there is no triable issue of material fact and that he is entitled to judgment as a matter of law. That is because of the general principle that a party who seeks a court's action in his favor bears the burden of persuasion thereon. [Citation.] There is a triable issue of material fact if, and only if, the evidence would allow a reasonable trier of fact to find the underlying fact in favor of the party opposing the motion in accordance with the applicable standard of proof. . . . [¶] [T]he party moving for summary judgment bears an initial burden of production to make a prima facie showing of the nonexistence of any triable issue of material fact; if he carries his burden of production, he causes a shift, and the opposing party is then subjected to a burden of production of his own to make a prima facie showing of the existence of a triable issue of material fact. . . . A prima facie showing is one that is sufficient to support the position of the party in question. [Citation.]" (Fns. omitted, see *Kids' Universe v. In2Labs* (2002) 95 Cal.App.4th 870, 877-878.) We review the trial court's decision to grant the summary judgment motion de novo. (*Johnson v. City of Loma Linda* (2000) 24 Cal.4th 61, 65, 67-68; *Sharon P. v. Arman, Ltd.* (1999) 21 Cal.4th 1181, 1188, disapproved on another point in *Aguilar v. Atlantic Richfield Co., supra,* 25 Cal.4th at p. 853, fn. 19.)

B. Triable Issues Of Material Fact.

Plaintiff contends she produced sufficient evidence to raise a triable issue of material fact as to whether fenfluramine caused her pulmonary hypertension, valvular heart disease, and coronary artery blockage. Plaintiff relies on Dr. Woods' opinions and observations he personally made of her cardiovascular system during the operation performed to bypass the blockage in her artery. As an alternative argument, plaintiff asserts defendant failed to meet its initial burden of showing there was no causal connection between fenfluramine use and pulmonary hypertension and valvular disease. This is because: defendant only argued below that there was no causal association between fenfluramine and plaintiff's cardiomyopathy and coronary artery disease; defendant did not dispute it is widely accepted in the medical community there is a causal connection between fenfluramine use and pulmonary hypertension and valvular heart disease; and the deposition testimony of the five treating cardiologists does not establish the absence of causation as to her heart condition due to the diet drugs.

Our Supreme Court has explained: "California law permits a person with 'special knowledge, skill, experience, training, or education' in a particular field to qualify as an expert witness (Evid. Code, § 720) and to give testimony in the form of an opinion (*id.*, § 801). Under Evidence Code section 801, expert opinion testimony is admissible only if the subject matter of the testimony is 'sufficiently beyond common experience that the opinion of an expert would assist the trier of fact.' (*Id.*, subd. (a).)" (*People v. Gardeley* (1996) 14 Cal.4th 605, 617.) Evidence Code section 801, subdivision (b) states: "If a witness is testifying as an expert, his testimony in the form of an opinion is limited to such an opinion as is: [¶] . . . (b) Based on matter (including his special knowledge, skill, experience, training, and education) perceived by or personally known to the witness or made known to him at or before the hearing, whether or not admissible, that is of a type that reasonably may be relied upon by an expert in forming an opinion upon the subject to which his testimony relates, unless an expert is precluded by law from using such matter as a basis for his opinion." (See *People v. Gardeley*, *supra*, 14 Cal.4th at p.

618.) Opinion based testimony by a properly qualified professional is required to show causation where a medical process is beyond common knowledge. (*Jones v. Ortho Pharmaceutical Corp.* (1985) 163 Cal.App.3d 396, 402; see also *Vandi v. Permanente Medical Group, Inc.* (1992) 7 Cal.App.4th 1064, 1071.)

In *Jennings v. Palomar Pomerado Health Systems, Inc.* (2003) 114 Cal.App.4th 1108, 1117, our colleagues in Division One of the Fourth Appellate District discussed medical opinion evidence on causation as follows: "It is undisputed that qualified medical experts may, with a proper foundation, testify on matters involving causation when the causal issue is sufficiently beyond the realm of common experience that the expert's opinion will assist the trier of fact to assess the issue of causation. [¶] However, even when the witness qualifies as an expert, he or she does not possess a carte blanche to express any opinion within the area of expertise. [Citation.] For example, an expert's opinion based on assumptions of fact without evidentiary support [citation], or on speculative or conjectural factors [citation], has no evidentiary value [citation] and may be excluded from evidence. [Citations.] Similarly, when an expert's opinion is purely conclusory because unaccompanied by a reasoned explanation connecting the factual predicates to the ultimate conclusion, that opinion has no evidentiary value because an 'expert opinion is worth no more than the reasons upon which it rests.' [Citation.]" (See *Kelley v. Trunk* (1998) 66 Cal.App.4th 519, 523-525.) In *Jennings*, the Court of Appeal continued: "[A]n expert's conclusory opinion that something did occur, when unaccompanied by a reasoned explanation illuminating how the expert employed his or her superior knowledge and training to connect the facts with the ultimate conclusion, does not assist the jury. In this latter circumstance, the jury remains unenlightened in how or why the facts *could* support the conclusion urged by the expert, and therefore the jury remains unequipped with the tools to decide whether it is more probable than not that the facts *do* support the conclusion urged by the expert. An expert who gives only a conclusory opinion does not *assist* the jury to determine what occurred, but instead supplants the jury by *declaring* what occurred." (*Jennings v. Palomar Pomerado Health Systems, Inc.*, *supra*, 114 Cal.App.4th at pp. 1117-1118 (original italics); see *Jennifer C.*

v. Los Angeles Unified School Dist. (2008) 168 Cal.App.4th 1320, 1332-1333.) The determination as to whether an opinion is speculative, and therefore insufficient to support a judgment, is as follows: "Where an expert bases his conclusion upon assumptions which are not supported by the record, upon matter which are not reasonably relied upon by other experts, or upon factors which are speculative, remote or conjectural, then his conclusion has no evidentiary value. [Citations.] In those circumstances the expert's opinion cannot rise to the dignity of substantial evidence. [Citation.]" (*Pacific Gas & Electric Co. v. Zuckerman* (1987) 189 Cal.App.3d 1113, 1135-1136; see also *Leslie G.* v. *Perry & Associates* (1996) 43 Cal.App.4th 472, 487; *Hyatt* v. *Sierra Boat Co.* (1978) 79 Cal.App.3d 325, 338-339; *Richard* v. *Scott* (1978) 79 Cal.App.3d 57, 63.)

In this case, Dr. Woods, who is a cardiothoracic surgeon, provided the following opinion, "[Plaintiff's] cardiomyopathy, valvular insufficiencies, pulmonary hypertension and blocked [left anterior descending artery] were caused by extended use of diet drugs." Dr. Woods's professional background includes the diagnosis and treatment of diseases of the thoracic cavity and heart. Moreover, according to Dr. Woods, Dr. Terry's initial diagnosis of cardiomyopathy of viral origin was incorrect. Dr. Woods's conclusion was based upon his personal observations of plaintiff's body during surgery. Dr. Woods explained how he reached his conclusion there was no viral related cardiomyopathy: "I looked at her heart and saw that her pericardium was completely clean and free and that there was no evidence of viral related pericarditis. I have operated on many people with histories of viral cardiomyopathy and this is not what you expect to see. There was no evidence of residual inflammation and scarring that you see when there has been viral related cardiomyopathy or heart failure." Thus, Dr. Woods's opinion that plaintiff did not suffer from cardiomyopathy of a viral origin was nonspeculative as it was predicated upon his review of her cardiovascular system.

Further, Dr. Woods's opinion was sufficient to raise an issue of material fact as to defendant's theory that diet drugs did not cause plaintiff's heart problems because she suffered from viral cardiomyopathy. Dr. Woods disagreed with Dr. Terry's initial diagnosis of viral cardiomyopathy. Each of plaintiff's treating cardiologists predicated

their opinions on Dr. Terry's initial diagnosis. But Dr. Woods's opinion was based on matter that is a type that may be reasonably relied upon by a surgeon, specifically his own observations of plaintiff's cardiovascular system during surgery. According to Dr. Woods: his observations included information which would not have been visible on an echocardiogram; he believed the presence of white lesions were consistent with literature showing valvular damage from diet drugs; and there was no yellow plaque which he would have expected from coronary artery blockage caused by cholesterol. Thus, we disagree with defendant that Dr. Woods's opinions are based on speculation. His opinion could not, at the summary judgment stage, be disregarded. We need not address plaintiff's argument defendant failed to sustain its initial burden of production on the causation issue.

IV. DISPOSITION

The judgment is reversed. Plaintiff, Diane Oakes, is awarded her costs on appeal from defendant, Wyeth, formerly known as American Home Products Corporation.

NOT TO BE PUBLISHED IN THE OFFICIAL REPORTS

TURNER, P. J.

We concur:

ARMSTRONG, J. KRIEGLER, J.